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ROCKY MOUNTAIN SPOTTED FEVER

BY

WM. COLBY RUCKER
Assistant Surgeon General, United States Public Health Service

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ROCKY MOUNTAIN SPOTTED FEVER.[1,2]

By W. C. Rucker, Assistant Surgeon General, United States Public Health Service.

HISTORY.

For over a decade Rocky Mountain spotted fever has been a problem of great interest to the physician, the zoologist, and the sanitarian. Its geographic limitation, seasonal prevalence, intimate association with wood ticks, and variation in severity in different localities combine to make it one of the most interesting and intricate disease problems which have arisen in our generation. It has a peculiar interest, because apparently it is confined to the American Continent, and it has therefore been considered appropriate to present a brief review of the progress in the study of the disease and to indicate the lines along which investigative and eradicative work should be carried in the future.

Although the disease has been known in Idaho and Montana since 1873, the first specific reference to it in literature is to be found in the report of the Surgeon General of the Army for the fiscal year ending June 30, 1896 (77). It is there stated that "the surgeon[3] at Boise Barracks referred in one of his monthly reports to the prevalence of spotted fever in the civil settlements in the neighborhood of the post. On being requested to give fuller particulars concerning this fever, he stated that as he had not seen any of the cases that occurred he had called upon his medical friends in civil life for information." These gentlemen (Drs. C. L. Sweet, W. D. Springer, R. M. Fairchild, L. C. Bowers, J. K. Dubois, D. W. Figgins, and H. Zipf) responded promptly and their reports constitute the first published accounts of Rocky Mountain spotted fever as a disease entity.

It was not until 1899, however, when Dr. E. E. Maxey, of Boise, Idaho, read a paper entitled "Some observations on the so-called spotted fever of Idaho" (32), before the Oregon State Medical Society, that the disease began to attract any widespread attention. This lucid paper expresses the opinion that spotted fever is a specific disease and gives an accurate description of its clinical manifestations.

In 1902, the then newly organized Montana State Board of Health selected for its first task the careful investigation of the disease, securing for this purpose the services of Drs. L. B. Wilson and

[1] Reprint from the Public Health Reports, Vol. XXVII, No. 36, Sept. 6, 1912.

[2] This paper originally appeared in the Military Surgeon, Vol. XXIX, No. 6, Dec., 1911, pp. 631–657, under the title, "The problem of Rocky Mountain spotted fever." As republished here the text and bibliography have been amended so as to cover the subject to the present time.

[3] Then Capt. (now Lieut. Col., Deputy Surg. Gen., retired) Marshall W. Wood, M. C., U. S. A.

W. M. Chowning, of the University of Minnesota. Their work, which was done in the Bitter Root Valley of Montana, constitutes the first serious laboratory study of the disease (12, 73, 74, 75, 76), and in a paper written July 1, 1902 (73), they suggested the rôle of the ground squirrel (*Citellus columbianus*) and the tick (*Dermacentor andersoni*) as host and vehicle of transmission, respectively. In the same year Surg. J. O. Cobb, of the United States Public Health Service, visited the Bitter Root Valley and wrote a description of the disease (13). Subsequent investigations have been made by Ashburn (5, 6, 7), Craig (7, 16), and Keiffer (26), of the Army; Anderson (1, 2, 3), Stiles (65–71, inclusive), Francis King (27), and McClintic (36), of the Public Health Service; and by several others, the most noteworthy among whom are the martyred Ricketts (42–55, inclusive) and his associates.

GEOGRAPHIC DISTRIBUTION.

The disease has been reported from nearly all the States in the Rocky Mountain group, California, Colorado, Idaho, Montana, Nevada, Oregon, Utah, Washington, and Wyoming, each having foci. Cases have also been reported from the District of Alaska. The geographic distribution of the disease is shown as follows in tabular form:

Geographic distribution of Rocky Mountain spotted fever.

States.	Locality.	Reporter.
Alaska	Klondike	Gwinn (21).
California	Calneva	Snow.[1]
Colorado	Carbondale	Braden (57).
	Rifle	Le Rosignol and Hotopp (57).
Idaho	Valleys of the Weiser, Payette, Boise, and Wood Rivers; north bank of the Snake River; southwestern Idaho.	Maxey (32).
Montana	West side Bitter Root Valley	Wilson (73) and Chowning; Anderson (1); Stiles (65); McClintic (36).
	Phillipsburg, Clinton, Camas Prairie	Anderson (2).
	Rock Creek, Blackfoot, Rattlesnake, and Lolo Valleys.	McCullough (37).
	Bridger	Gates (2, 67).
	Livingston	Alton (65).
Nevada	Quinn River Valley	Kendall (2).
	Paradise Valley, Winnemucca, Fort McDermitt, Reno.	Robinson (57).
Oregon	Burns	Geary (19).
	Lakeview	Steiner (57).
	Merrill	Patterson (57).
Utah	Cedar Valley, Fairfield, Cedarfort	Noyes (57).
	Heber City	Wheritt (57).
Washington	Moses Lakes, Douglas County	Smith (62).
Wyoming	Thermopolis, Myersville, Shoshone River	Gates (2, 67).
	Crow Creek	Kieffer (26).
	South Pass, Fort Fetterman, Fort Steele, Cheyenne	Robinson (57).
	Cody, Meeteese	Bradbury (76).

[1] Personal letter.

Data regarding the prevalence of Rocky Mountain spotted fever in the known infected localities is very sparse except in Montana and Idaho. In the latter State Dr. Edward E. Maxey, of Boise, collected data on 380 cases which occurred during 1908. The following table shows the occurrence of the disease in the Bitter Root

Valley from 1885 to 1911, inclusive, representing data collected by Wilson and Chowning, Anderson, Stiles, and McClintic:

Human cases of Rocky Mountain spotted fever in the Bitter Root Valley of Montana.

Year.	Cases.	Deaths.	Case fatality rate.	Year	Cases.	Deaths.	Case fatality rate.
			Per cent.				*Per cent.*
1885	1	1	100	1900	12	9	75
1886	1	1	100	1901	14	10	71.4
1887	0	0	0	1902	21	15	71.4
1888	3	1	33.3	1903	14	9	64.2
1889	3	3	100	1904	11	9	81.8
1890	1	1	100	1905			
1891	6	4	66.6	1906			
1892	3	1	33.3	1907			
1893	4	2	50	1908	12	5	41.6
1894	0	0	0	1909	28	13	46.4
1895	3	3	100	1910	19	14	73.6
1896	6	6	100	1911	16	6	37.5
1897	6	5	83.3	1912	1	1	100
1898	3	2	66.6	(1)	4	2	50
1899	23	14	60.8				

[1] Year not definitely known.

It might be well to point out at this time the necessity for the careful collection of data regarding the occurrence of cases in the various infected States. Rocky Mountain spotted fever certainly should be put on the list of reportable diseases.

SYMPTOMS IN MAN.

Passing now to the consideration of the clinical aspects of the disease in man, Maxey's definition of the disease may be modified to read, "Rocky Mountain spotted fever is an acute, endemic, febrile disease, occurring chiefly during the summer months, transmitted by the bite of the tick, and characterized clinically by a continuous moderately high fever, severe arthritic and muscular pains, and a profuse petechial or purpuric eruption in the skin, appearing first on the ankles, wrists, and forehead, but rapidly spreading to all parts of the body."

After an incubation period varying from 3 to 10 days, usually 7, during which the patient may feel indisposed and complain of ill-defined sensations of cold, nausea, and weariness, there is a frank chill. If seen at that time the patient will generally complain of pain and soreness in the muscles, bones, and joints, especially in the lower lumbar region. Severe occipital headache and photophobia are frequent symptoms and the face may appear flushed and swollen. Epistaxis commonly occurs and constipation is the rule. The severity of the symptoms varies in individual cases and is less severe in Idaho than in Montana.

Upon examination the face is apt to be flushed, and the conjunctivæ congested and yellowish. The tongue is covered centrally with a heavy white coat, while its tip and edges are bright red. A slight bronchitis may exist, and the urine is scanty and may contain small amounts of albumin and a few casts. Prior to the initial chill there

may be a little afternoon fever, but with the chill there is an abrupt elevation of temperature and on the successive days there is an evening rise with slight morning remissions. At any time from the eighth to the twelfth day, usually the tenth, the fastigium is reached, when, if the patient is to recover, a fall by lysis takes place, the curve reaching subnormal from the fourteenth to the eighteenth day and remaining so for three or four days. In certain of those cases which do not recover there is a continuous fever of 105° F. or higher. In other cases there is a sharp drop in the temperature curve, followed by a sudden rise just before death.

The pulse is very rapid and apt to be thready. There is a progressive decrease in the erythrocytes and hæmoglobin. A leucocytosis with considerable increase in the large mononuclears occurs.

The respiration rate is increased in proportion to the pulse. An initial bronchitis is not uncommon and hypostatic pneumonia sometimes occurs.

Usually on the third day (sometimes on the fourth) the eruption appears on the wrists and ankles, first as a macular roseola, which, as it spreads to the arms, legs, forehead, back, chest, and abdomen, in the order named, becomes papular and may terminate in indefinite blotches or petechiæ which may become large ecchymotic spots. In severe cases even the palms, soles, and scalp may be invaded. From 12 to 48 hours are required for the rash to reach the maximum. The macules vary in size from a pin point to a split pea and are bright red except when the case is unusually severe, when they are dark purple. Not infrequently they assume this color after death. It was this sign which caused the earlier cases to be called "the blue disease" or "black measles." The macules disappear readily on pressure, rapidly to return—the papules do not disappear on pressure until the patient is progressing to recovery. With the fall in the fever the eruption begins to fade, but for a considerable time after recovery it may reappear as a subcuticular mottling after free perspiration or a warm bath. Cases have been reported in which there was no exanthem. Late in convalescence there is a generalized desquamation. Gangrene of the ears, fauces, fingers or toes, scrotum, penis, or entire pudenda may occur as distressing sequelæ. Hæmatogenous jaundice usually occurs, and in addition the face may have a bloated appearance, erasing the lines of expression and giving it a stupid look.

The teeth are covered with sordes early and the tongue is coated throughout the disease. This coating is at first white, but later it becomes light yellow and finally dirty brown. The mouth is dry and cracked. Constipation, sometimes extreme, exists throughout the disease. Initial nausea, which may extend throughout the disease, is not uncommon. There is splenic and hepatic enlargement.

The urine is high colored, acid, and reduced in amount. Albumin and granular, hyaline, and epithelian casts are found in about 50 per cent of the cases. Hemoglobinuria almost never occurs.

The mind is usually clear throughout the disease. During the period of invasion there may be restlessness and insomnia owing to the attendant pain in the bones and muscles. Later this is absent. Kernig's sign is not found. Ocular symptoms are very rare.

SYMPTOMS IN ANIMALS.

The reactions which occur when laboratory animals are inoculated with the disease are fairly constant. Guinea pigs when given 0.5 c. c. to 5.0 c. c. of defibrinated infected blood, serum, or washed corpuscles, subcutaneously or intraperitoneally, present a rise of temperature after an incubation period varying from two to five days. From the fifth to the seventh day the temperature may reach 107.6° F. Coincident with the fastigium, the scrotum and testicles become swollen and œdematous, and subsequently the overlying skin of the pudenda becomes the seat of hypodermic hemorrhages of varying size and outline. Vulvar changes occur in female guinea pigs, but are less constant. The soles of the feet and the ears are red and congested, and if the animal be depilated, reddish macules may be observed on the dorsal and lateral aspects of the body. Emaciation is rapid, and death usually occurs from the seventh to the eleventh day. Recovery, when it takes place, is gradual and may be accompanied with scrotal sloughing, followed by deforming cicatrix formation. There is desquamation of the soles of the feet, and the ears become dry and brittle, subsequently dropping off, leaving a short, thickened, irregular stump. The animal is emaciated and may not regain its normal weight for several weeks. When the disease is transmitted by ticks the signs are much the same, except that there may be areas of necrosis and patchy alopecia at the points where the ticks attached.

In monkeys (*Macacus rhesus*) the disease produces cyanosis of the face and ears, a skin eruption varying from an erythema to a macular and petechial marking distributed over the external aspects of the arms, legs, buttocks, and back. The scrotum and penis are enlarged and hæmorrhagic.

The rabbit (*Lepus sp.*) is mildly susceptible to the virus, but in far less severe form than in guinea pigs and monkeys. After an incubation period varying from three to six days, the temperature reaches 104° F. and falls by lysis. Aside from congestion of the scrotum no marked anatomical changes have been recorded. The susceptibility of the various domestic animals and the mammals of the infected zone will be discussed elsewhere.

PROGNOSIS.

In the Idaho cases the prognosis seems to be very favorable, as a rule the case fatality rate averaging less than 4 per cent. The disease is far more lethal in Montana, and there the case fatality rate averages close to 75 per cent, although in some years it has fallen as low as 33.3 per cent. Death may occur as early as the third or as late as the eighteenth day of the disease. In general, if the patient survive the tenth day, the prognosis is far more favorable. Continuously high fever or a sudden drop in temperature are grave signs, as is also delirium or loss of consciousness.

GROSS PATHOLOGY.

The pathological changes are not extreme, but they are fairly characteristic. In man rigor mortis usually appears early and is intense. The skin changes observed at necropsy are practically the

same as those seen ante mortem and include the small wounds the result of tick bites. Icterus is constant and cutaneous hemorrhages of varying sizes and shape are usually seen. In the Idaho cases gangrene of the fauces, tonsils, and palate, and of the scrotum, penis, and vulva have been noted. Aside from occasional hypostatic congestion and a rare pneumonia, the respiratory apparatus is usually normal. Epicardial hemorrhages over the ventricles were constantly found in Anderson's cases (2). The heart muscle is flabby, soft, and pale. The right heart is usually full of firmly coagulated blood, while the left heart is contracted and empty. The spleen is usually enlarged to three or four times its normal weight, is dark purple, soft, and very friable. The liver is enlarged and shows cloudy swelling and fatty degeneration. The pancreas is about twice its normal weight. The intestines may show submucous hemorrhages. Le Count (28) notes the enlargement of the superficial and visceral lymph glands. The kidneys are usually enlarged and present subcapsular and pelvic hemorrhages. The other abdominal viscera are not markedly affected. The changes in the nervous system are not constant enough to be of value in the post-mortem diagnosis of the disease.

In guinea pigs the pathological changes noted include coagulation necrosis about the site of inoculation; enlargement of the superficial lymph glands, with central hemorrhages and degeneration; splenic and hepatic changes similar to those observed in man; enlargement of the suprarenal bodies; localized hemorrhages with necrosis of the pudenda; and gangrenous changes of the ears. The lesions in monkeys are practically identical with those observed in man.

MICROSCOPIC PATHOLOGY.

The microscopic "changes are of two sorts, those connected with the occlusions of vessels and the more diffuse lesions affecting entire groups of organs. The diffuse changes are hyperplasia of lymphoid tissues and cloudy swelling and acute fatty changes in organs commonly the seat of such lesions in acute infectious diseases. The focal lesions are more varied in their nature, since they include not only the processes leading up to the occlusion of vessels, but the results of such obstructions, necrosis in different degrees and the hemorrhages responsible for so many of the clinical and gross anatomic features of the disease as well as for the name 'spotted fever' (28)." The minute changes have been made the subject of a careful study by Le Count (28) to whose article the reader is referred.

TREATMENT.

Many methods of treatment have been advised and employed in the attempt to cure this disease. They run the gamut of the Pharmacopœia from sage tea to quinine and they have returned to that tacit admission of ignorance "good nursing and symptomatic medication." Ricketts (53, 55) has produced a protective (and if given very early, and in large doses, curative) serum which Heinemann and Moore (22) have attempted to concentrate. The number of cases in which it has been used is too small to judge of its efficacy. Dr. Karl Kellogg, of Stevensville, Mont., and Dr. J. Wilson Reed,

of Victor, Mont., have each used sodium caccodylate with apparent success in a single case. McClintic (36) treated monkeys infected with Rocky Mountain spotted fever with sodium caccodylate, salvarsan, and urotropin. None of these agents seemed to exert a beneficial effect on the disease. Until we are better informed as to the etiology of the disease all attempts at its cure must be empirical and groping.

ETIOLOGY.

When we attempt the consideration of the etiology of this disease we are in a certain measure entering a terra incognita. As noted by Maxey (32), in his original paper, spotted fever is a "place" disease, being definitely limited to a certain locality—for example, to a single side of a valley. It is also rather sharply limited to a definite season of the year, usually to the months of March, April, May, June, and July. It attacks all ages and both sexes, although the greater number of cases have occurred in males between 30 and 40 years of age. Persons whose occupations take them into the wooded foothills seem more liable to the disease; therefore, the bulk of the cases have occurred in lumbermen, miners, prospectors, ranchers, and sheepherders, and bridge builders, carpenters, civil engineers, and others concerned in railroad construction work. It is apparently noncontagious, more than a single case rarely occurring in a given household at the same time. It has been impossible to incriminate water or food of any kind as the vehicles of infection, although when Maxey presented his first paper he suggested that the drinking of snow water might be the means of receiving the disease.

THE TICK HOST.

Wilson and Chowning in their original report (73) suggested the hypothesis that the wood tick (*Dermacentor andersoni*) acted as the transmitting agent and offered in support of this theory several facts which may be thus summarized:

1. The appearance of the disease is coincident with the period of activity of the wood tick.
2. The disappearance of the disease is coincident with the disappearance of the wood tick.
3. The limitation of the disease in a certain locality suggests the conveyance of the germ to man by a temporary parasite "traveling slowly and not widely and which is not carried far by the wind. The tick answers this description."
4. The great bulk of patients give a history of having been bitten by ticks prior to their illness.
5. Mosquitoes may be eliminated from the problem because their appearance and disappearance does not coincide with that of spotted fever; because of their lack of geographic limitation; and because they would be more apt to bite and thus infect a greater number in a given family. Bedbugs and fleas are omnipresent and perennial; spotted fever is not.

Cobb (13), Anderson (1, 2), Westbrook (57), and R. W. Smith (57) coincided in this view, but Stiles (65) was "unable to confirm this hypothesis." Ashburn (5) reached the same conclusions as Stiles.

In 1906, King (27) succeeded in transmitting the disease from one guinea pig to another guinea pig by an adult male tick and Ricketts (42–47) was able to similarly transfer the infection by an adult

female tick. In the following year, 1907, Ricketts (46) demonstrated that infected ticks exist in nature on the west side of the Bitter Root Valley of Montana and by their bites he reproduced the disease in guinea pigs. He further showed that the larvæ and nymphs and both adult male and female ticks infected by feeding on an infected animal may transmit the disease to normal susceptible animals; that larvæ and nymphs may acquire the disease in a similar manner and that they are capable of transmitting it in their subsequent stages of development; that infected females may transmit the disease to their young through their eggs; that the infection is generalized in the body of infected ticks; that the virus remains active in the body of the nymphal tick; that infected ticks are infective as long as they live and will bite. From the foregoing it may be deduced that the tick is the disseminator of the casual agent of the disease in nature. As a final and clinching proof, McCalla (35) removed a tick from a man suffering with the disease and, with their consent, infected a man and a woman by its bite.

Since it has been proven that the disease exists in ticks in nature, it is to be expected that the distribution of the disease is the same as the distribution of the dermacentor. This has been made the subject of a study by Bishop (9) and while Rocky Mountain spotted fever has not been reported from the entire life zone of this tick, with the exception of the cases which occurred in the Klondike, the disease has not been found outside the area which the tick infests. This includes the northern part of the Rocky Mountain region in the United States, and the river valleys and sagebrush plains to the west, the western corner of South Dakota, almost the entire States of Montana, Wyoming, and Colorado, the northern portion of New Mexico, Utah, and Nevada, all of the State of Idaho, the eastern half of Washington and Oregon, and the northeastern corner of California. It also occurs in southern British Columbia and eastern Alberta.

There has been more or less discussion regarding the taxonomy of this species, but that is a question for zoological nomenclaturists which need not be considered here, and it should be borne in mind that Maver (31) has transmitted the disease by three other species, *Dermacentor marginatus* (Utah), *Amblyomma Americanum* Linnæus (Missouri), and *Dermacentor variabilis* (Mass.). It may be of profit, however, to describe briefly the commonest form (*D. andersoni*) and to outline its life history.[1]

THE ANATOMY OF TICKS.

Ticks, superfamily Ixodoidea, order Acarina, class Arachnida, represent the giant mites. Anatomically a tick may be divided into a head, rostrum, or capitulum, and a body. The capitulum consists of a neck which connects it with the body; a hard, usually quadrangular portion called the base, which presents two porose areas and supports the palpi, which are composed of four segments, the hypostome, and elongated structure in symmetrical halves, which are covered with minute recurving teeth, and the mandibles or biting apparatus. The body is more or less ovoid in shape and

[1] The writings of Stiles (69–70) and Ricketts (42–56) have been freely drawn upon in the preparation of this description. For a more technical consideration of the subject the reader should consult Bull. 62, U. S. Public Health and Marine-Hospital Service, Hyg. Lab., 1910.

varies greatly in form, color, outline, and structure in the different species and at different periods of development. The body is divided for purposes of description into a dorsal surface, a ventral surface, and anterior, posterior, and lateral margins. The dorsal surface presents a hard, chitinous plate, marked by two longitudinal grooves. This is called the scutum and is smaller in the female than in the male. The eyes are seen at each lateral margin of the scutum, and on each side of the median line, near the third and fourth legs, are small, oval, chitinous structures called the dorso-submedian porose plates. Along the posterior margin of the body are the postero-marginal festoons, 11 in number. The ventral surface presents for examination the genital pore, situated between the coxæ of the first three pairs of legs; the anus, similarly situated in the median line but behind the posterior pair of legs; and the stigmal plates placed laterally just behind the fourth pair of legs. The anterior, posterior, and lateral margins vary in the different species. The legs are four in number on each side and each is segmented into a cova, trochanter, femur, patella, tibia, and tarsus. Both the dorsal and the ventral surfaces present grooves, pits, hairs, and spines which are of value in distinguishing the various species.[1]

THE DERMACENTOR ANDERSONI.

The *Dermacentor andersoni* Stiles (1905), male is oval, narrow in front, broad behind, with scutum variegated brown and white. Anteriorly there is an elliptical area, called the pseudoscutum, limited by a white border and possessing two lateral brown stripes, with a median brown stripe or spots between them. Behind this there are 4 brown stripes arranged in a curve, open anteriorly. Posterior to these there are usually 5 brown stripes, 1 central and 2 on each side. Elsewhere the whole dorsum is speckled with small brown dots. The 11 festoons of the posterior border are roughly quadrangular in outline and consists of a white area with 1 brown spot and small brown specks. On the ventral surface, it is noted that the first coxæ arise by two roots, bidentate, while the others arise by a single spine. The fourth coxa is very large, being two or three times the size of the third. Opposite the second pair of legs is the genital aperture. The stigmal plates are somewhat comma shaped.[2]

The noneengorged female is about the same size as the male, 5 by 2.5 mm. The body is oval and broader posteriorly than anteriorly. The scutum extends as far back as the third pair of legs and is marked like the corresponding portion of the scutum of the male. There is a dorsal marginal groove and three longitudinal grooves. Eleven festoons on the posterior margin. The genital aperture on the ventral surface is opposite the second coxa, and from it the genital grooves run backward, diverging laterally behind the fourth coxa and ending between the second and third external festoons. There is a short naomarginal groove. The replete female is about 16 by 10 mm. and deep brown or slate color.

[1] For a description of the internal anatomy see Christophers (S. R.), The Anatomy and Histology of Ticks. Calcutta, 1906.

[2] See Stiles's "The taxonomic value of the microscopic structure of the stigmal plates in the tick genus Dermacentor." Bull. 62 U. S. Public Health and Marine-Hospital Service, Hyg. Lab., 1906.

The adult male and female feed in common on various mammals, and it is during this time that copulation and fertilization take place. The female continues to feed for several days after fertilization until she has become a slate-colored, swollen ovoid body. This increase in size is due to the ingestion of blood from the host and the enlargement of the ovaries and beginning formation of hundreds, or even thousands, of minute eggs. After complete engorgement, the female drops from the host and after a resting period of about two weeks begins oviposition. To accomplish this the head is bent ventrally until the capitulum rests on the edge of the genital opening. At the same time there is protruded from beneath the scutum a delicate white gelatinous membrane which terminates in two delicate cones covered with an adhesive secretion. The extrusion of this membrane covers the head, and as the two small sticky cones reach the genital orifice the egg is expelled onto them. The membrane is then withdrawn and the head extended, the egg resting on the front of the scutum. In this way an adherent mass of eggs gradually forms in front of the tick.[1] Unless observed closely, it appears as though the eggs were being extruded from beneath the scutum. As this process continues the tick begins to shrivel and at the end of oviposition it dies. The number of eggs deposited varies from several hundred to three thousand.

The length of time before the eggs begin to hatch depends on the surrounding temperature. In the summer months it is from 30 to 50 days, but in the cold season it may be delayed for several months. From the egg appears the larval form of "seed-tick" stage. These are minute specks, which are first pale and soft, and later become covered with a hard brown coating. They have six legs and are without genital and spiracular orifices. They are seen in nature in clumps on blades of grass or twigs, where they wait with outstretched legs for passing mammals. Having attached themselves to a warm-blooded host, they feed to engorgement in about six days. During this time the original bulk is increased many fold, reaching about the size of a head of a pin. The color, which depends on the character of the food taken, blood or serum, varies from light pink to dark brown. Having fed to surfeitment, the tick drops off and lies dormant for about four weeks prior to moulting. Here again the time varies, being as short as two weeks and as long as two months. Unless the larvæ secure food within two or three weeks after hatching they die.

After the larval skin is cast, the nymph, having four pairs of legs and spiracular orifices but no genital aperature, emerges. It is about 1.5 mm. in length and is at first slightly yellow in color, but after feeding becomes brownish black. Again it awaits a host, and having secured one feeds from four to eight days, becoming greatly enlarged—4 by 2 mm.—and eventually dropping off as in the previous stage of its development. It does not immediately reenter the dormant state, but may be active for a period varying from two to four weeks. This is apparently influenced by the atmospheric temperature. Eventually, however, it becomes quiescent and lies dor-

[1] See Braun (M.), The Animal Parasites of Man. Wm. Wood & Co., N. Y., 361.

mant for about a month, while the metamorphosis into the adult is being completed. When this is completed a second moult takes place and it emerges from the snowy white shell a mature tick, with genital orifices and the secondary sexual characteristics typical of the male or female. The adults now attach themselves to a warm-blooded host, and after a time copulation, fertilization, and oviposition take place, and the cycle is recommenced. It is believed that the tick produces but one brood a year. It may be noted also that Cooley (15) quotes W. V. King, of the Montana Agricultural College, as suggesting the hypothesis that the life cycle of this tick (*D. andersoni*) is two years. Additional experimental evidence seems needful to prove this.

MAMMALIAN HOSTS OF THE D. ANDERSONI.

It is, of course, important that we know what animals act as the hosts for these ticks. This has not only a direct bearing on tick control, but it may also lead us to the discovery of the animal which acts as the intermediary host for the virus, provided, of course, that such is necessary for the perpetuation of the disease. Data has therefore been collected bearing on this important aspect of the question.

Animals on which the D. Andersoni has been found, their susceptibility to Rocky Mountain spotted fever, and the stage of development of the tick.

Animal.	Susceptible.	Adult.	Nymph.	Larvæ.
Mule deer (*Odocoileus hemoinus*)	?	+	+	
Elk (*Cervus canadensis*)	?	+		
Mountain goat (*Oreamnos montanus*)	?	+	+	
Mountain sheep (*Ovis canadensis*)	?	+		
Pine squirrel (*Sciurus hudsonicus richardsoni*)	?		+	+
Yellow-bellied chipmunk (*Eutamias luteiventris*)	+		+	+
White-bellied chipmunk (*Eutamias quadrivittatus umbrinus*)	+		+	
Columbian ground squirrel (*Citellus columbianus*)	+		+	+
Side-striped ground or rock squirrel (*Callospermophilus lateralis cinerascens*)	+		+	+
Woodchuck (*Marmota flaviventris*)	+	+	+	+
White-footed mouse (*Peromyscus maniculatus artemisiae*)	0		+	
Wood rat (*Neotoma cinerea*)	+		+	
Meadow mouse (*Microtus modestus*)	0		+	
Porcupine (*Erethizon epixanthum*)	?	+		
Rock cony or rabbit (*Ochotona princeps*)	?	+	+	+
Snowshoe rabbit (*Lepus bairdi*)	?	+	+	
Cottontail rabbit (*Sylvilagus nuttali*)	+		+	
Coyote (*Canis lestes*)	0	+		
Badger (*Taxidea taxus*)	0	+		
Weazel (*Putorius arizonensis*)	+	+		
Black bear (*Ursus americanus*)	?	+		
Marten (*Mustela c. origenes*)	?	+		
Dog (*Canis familiaris*)	?	+		
Cow (*Bos taurus*)	0	+		
Horse (*Equus caballus*)	+	+		
Sheep (*Ovis aries*)	0	+		
Swine (*Sus scrofa*)	0	+		

In studying this aspect of the question Ricketts endeavored to determine the following points with regard to the ground squirrel, the ground hog, the rock squirrel, the chipmunks, and the mountain or wood rat:

1. Is the animal susceptible to the disease by inoculation?
2. Is the animal susceptible to the disease through tick bites?
3. Can the "tick cycle" be completed on the animal (i. e., receive the disease through ticks and subsequently infect another tick)?
4. Has the animal the disease in nature?

The results of his experiments may thus be tabulated:

Susceptibility to Rocky Mountain spotted fever.

Species.	Inoculation.	By bites.	Tick cycle.
Ground squirrel	+	+	+
Ground hog	+	+	+
Rock squirrel	+	+	+
Chipmunks	+	+	?
Mountain rat	+	?	

MAMMALIAN HOSTS FOR THE DISEASE.

The question of the occurrence of the disease among mammals in nature has not yet been solved, although considerable work has been and is being done upon it. It is an enormous problem and involves the examination of great numbers of live wild animals, the determination of their immunity to spotted fever and the inoculation of their blood into laboratory animals to find out if they (the wild animals) have the disease in acute form. These points must be settled if we would discover the animal which perpetuates the disease.

During the summer of 1912, shortly before his heroic death, McClintic discovered an infected tick on the body of a Rocky Mountain goat (*Oreamnos montanus*) and it may be that the direction of the search for the mammalian host has been pointed out thereby. Certain it is that goats and spotted fever abound on the western side of the valley, while on the eastern side, where there is no fever, there are no goats. Also those valleys on the western side of the main valley, which have no goats, also have no fever. Furthermore it is stated that in those small valleys in which formerly large numbers of Angora goats (*Capra angorensis*) were kept, there was much fever, but that since the removal of these animals the disease has disappeared. These facts appear significant and worthy of investigation.

THE VIRUS.

With regard to the virus itself, even less is known. Wilson and Chowning (73) described certain ovoid intracorpuscular bodies in both fresh and stained blood taken from persons suffering with spotted fever. Anderson (1, 2, 3) agreed with them that this organism, which they named "piroplasma hominis," was "very probably the cause of spotted (tick) fever." Ashburn (5) and Stiles (65–67) failed to confirm this view and the latter stated that "indications are not lacking, that at least some of the stages of the supposed piroplasma hominis consist in reality of vacuoles, blood platelets, blood dust, artifacts, and tertian malaria parasites.' Maxey, Simon, and Cole found no piroplasms in the blood of cases in Idaho, and neither Ricketts nor Kieffer secured evidence which convinced them of the existence of the parasite of Wilson and Chowning. Francis was unable to find the organisms and Craig expressed the belief that the supposed piroplasms were merely degenerated erythrocytes.

Ricketts (54) described a pleomorphic bacterium which he believed might bear a casual relation to the disease. The form which he

most commonly found was that of "two somewhat lanceolate chromatim-staining bodies, separated by a small amount of eosin-staining substance." These bodies were found in the blood of men, animals, and ticks infected with the disease, and seemed to stain best with Giemsa's stain. The eggs of infected ticks from both Montana and Idaho were found to contain large numbers of minute bipolar-staining bacilli, apparently in various developmental stages. These organisms are found in large numbers in the salivary glands, alimentary sac, and ovaries of infected females, but they have not been found in the viscera of noninfected male and female ticks. This bacillus has not been grown on artificial media. Ricketts suggested for it the name "the bacillus of Rock Mountain spotted fever." He found that it does not agglutinate with low dilutions of immune human serum, but agglutinates distinctly in dilutions of 1 to 10, 1 to 20, and 1 to 40. It does not agglutinate with the higher dilutions. Normal human serum, on the contrary, caused clumping in a dilution of 1 to 1, a very slight agglutination in 1 to 10, and none at all in the higher dilutions. In immune guinea pig serum complete agglutination was present in dilutions up to 1 to 160. Normal guinea pig serum produced practically no agglutination at all. Until further proof is brought forward the casual rôle of Rickett's bacillus must be considered as unproven, although there are many facts which strongly suggest it as the infecting agent.

Whatever may be the cause of Rocky Mountain spotted fever, however, several facts regarding it have been clearly established. It is nonfiltrable; its infectiousness is largely destroyed by grinding it in a ball mill. At 50° C. the infectiousness of the virus is destroyed in 25 or 30 minutes. Infected blood kept in the ice compartment of a refrigerator loses its infectiousness after 15 or 16 days. The pathogencity of the virus is lost between 24 and 48 hours after complete desiccation. It may be kept alive by passage through guinea pigs, monkeys, rabbits, and ground squirrels (*Citellus columbianus*). The virus is present in the body fluids generally. It produces a rather high degree of immunity.

Whether the organism of Rocky Mountain spotted fever be a protozoon or a bacterium, the fact that it is transmitted to man by the bite of the tick suggests the necessity of some host mammal for the perpetuation of the disease. It is true that in malaria, the protozoon disease type, the hemaneba has but two life cycles, but it is apparent that the opportunities for biting man which the short-seasoned tick possesses are infinitely less than those of the Anopheles. Neither is the disease analogous in its etiology to yellow fever nor to the tick fever of Africa, because both the *Stegomyia calopus* and the *Ornithodorus moubata* are essentially domestic in their habits, whereas the *Dermacentor andersoni* comes in contact with man only accidentally. Also the feeding habits of this species would preclude man from being anything but an accidental host. Hereditary transmission to the eggs of infected females explains how the disease may be kept alive from one spring to the next, but would not account for the perpetuation of the disease, since not more than 50 per cent of the females transmit the disease to their young. At this ratio, when it is considered that on account of the many accidents of nature, only a small percentage reach maturity and only a small number of these become

fertilized, it would be a matter of a short time only until the disease became extinct from natural causes.

The domestic and wild animals remain to be considered as possible hosts. This has already been discussed with regard to the wild animals and among them the search has been narrowed down to a few small mammals. Among the domestic animals, the horse has a relative resistance to the disease, while the ox, sheep, and the fowl have a demonstrated resistance. Cats and dogs may possibly play a part in keeping the virus alive, but it is extremely improbable. The larger wild mammals such as deer, elk, bear, etc., wander over wide stretches, certainly into districts where spotted fever does not prevail and are never in continuous close proximity to human dwellings. For the present they may be eliminated from the problem. From the list of larger wild mammals the mountain goat should be excepted. Its range is pretty well confined to the western side of the valley and the Clearwater country beyond. As has been said before, this species should certainly be taken into account.

The white-footed mouse, meadow mouse, coyote, and badger are apparently not susceptible to the disease by inoculation, and since rabbits are infected with some difficulty the rock cony, snowshoe rabbit, and cotton-tail rabbit may be dropped from consideration. Of the animals which remain, the ground squirrel, the ground hog, the rock squirrel, the chipmunk, the mountain rat, and the weazel seem to be the most important. On account of the prevalence of the ground squirrel (*Citellus columbianus*) in the infected zone this species has been regarded with the greatest suspicion, although it is not impossible that several other species may also act as hosts for the virus. The small mammals mentioned certainly enter the problem as sources of food supply for the tick.

PROPHYLACTIC AND ERADICATIVE MEASURES.

We are dealing then with a disease whose cause and intermediary host are unknown, but whose disseminating agent we know and can attack. For the present the tick must be the focal point of all prophylactic and eradicative measures. Inasmuch as domestic stock furnish a convenient supply of food for the tick during its various developmental stages, and that the female tick is fertilized during feeding, the killing of ticks on cattle, horses, and sheep is of great importance. This is accomplished by dipping the tick infested animal in crude oil or some of the well-recognized arachnicides, such as cresylic acid, the arsenic salts, or extract of tobacco. This should be done at frequent intervals from March 1 to July 15 and should include all the animals in the infected zone. If for any reason it is not desired to dip any particular animal the ticks may be picked off every four or five days and destroyed.

The clearing and burning of land is a useful measure. This kills the tick directly, and on account of the exposure to the bright sunlight prevents the hatching of the eggs. The feeding of cattle in tick-free lots, if done universally, would prevent any increase in the number of ticks. The alternation of pasture has been found of service in combating Texas tick fever and might be of use in the eradication of Rocky Mountain tick fever.

The slaughter of the small mammalian hosts has long been considered a logical measure. This applies particularly to the ground squirrels, which are not only a perineal source of food and habitation for the larval and nymphal ticks, but which may possibly prove to be the intermediary host for the virus. The methods to be used in the destruction of these pests were fully described in a previous paper.[1]

Personal prophylaxis is, of course, very important and includes the wearing of tick-proof clothing by all persons entering the infected zone during the season of tick prevalence and the careful daily search of the body for ticks which may have attached themselves and has escaped notice. Ticks should be removed as soon as discovered. In doing this the tick should be given a gentle pull, lest the head be torn off and left in the skin to make a very annoying infection nidus. Another way to remove the tick is to grease it. This closes its respiratory spiracles and causes it to loosen its hold and drop off. When attached very firmly and for some time they may be pried off by a needle thrust into the skin immediately beneath the tick's head. After the removal of the tick the wound should be cauterized with a toothpick dipped in 95 per cent carbolic acid. If there is any suspicion that the tick was received in the zone of infection the bitten person should be given a protective dose of Ricketts's serum.

[1] Rucker (W. C.), Enzootic Plague in the United States. The Military Surgeon, 1911, xxviii, 1–6.

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